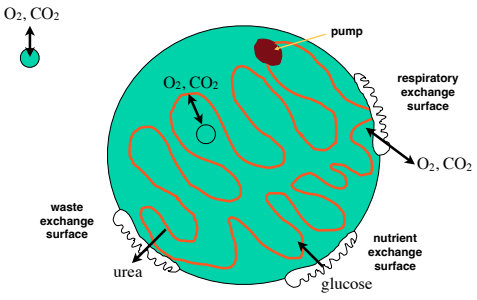
Human Phys PCB4701

Heart 1
Fox Chapter 13 part 1
(Chapter 12.6 Cardiac Muscle)

Anatomy of The Heart
Cardiac Cycle
Cardiac Action Potential and ECG

Circulatory System: Active Pumping
to transport gases from respiratory surface to tissues



Atrium
receives incoming blood, passes it to ventricle

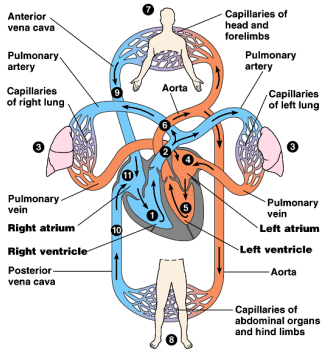
Ventricle
more muscular pump sending blood to a separate circulation (either pulmonary circulation (lungs) or systemic circulation).

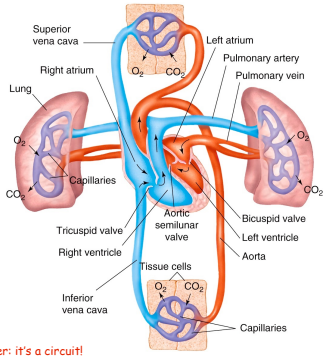
Arteries (arterial blood)
vessels carrying blood from heart towards the capillaries.
Thick muscular walls to keep pressure up.
High in oxygen (except for pulmonary arteries).

Veins (venous blood)
vessels carrying blood from capillaries back to heart. Very thin flabby walls with low pressure, but have one-way valves to prevent blood from backing up. Low in oxygen (except for pulmonary veins).

Capillaries
very small vessels (one blood cell wide) that perfuse all the tissues.

Figure 42.4 The mammalian cardiovascular system: an overview





remember: it's a circuit!

Figure 13.10

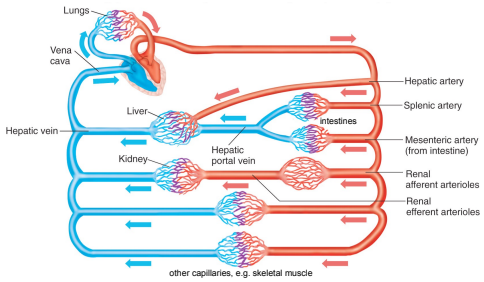


Figure 14.17

William Harvey, MD

1578-1657
Physician to Kings James I and Charles I

Author of ***de Motu Cordis et Sanguinis***
(An Anatomical Exercise on the Motion of the Heart and Blood in Living Beings), 1628

"This book is important both for the discovery of the complete circulation and for the experimental, quantitive and mechanistic methodology which Harvey introduced. He looked upon the heart, not as a mystical seat of the spirit and faculties, but as a pump analyzable along **mechanical lines.**

He observed that with each beat two ounces of blood leave the heart; so that with 72 heart beats per minute, the heart throws into the system 540 pounds of blood every hour. Where could all this blood come from? The answer seems to be that it is the same blood that is always returning. Moreover, the one-way valves in the heart, like those in the veins, indicate that ... the blood goes out to all parts of the body through the arteries and returns by way of the veins. The blood thus makes a **complete closed circuit**."

described pulmonary vs. systemic circulation, cardiac cycle, pacemaker cells, arteries vs. veins...(but couldn't see capillaries)

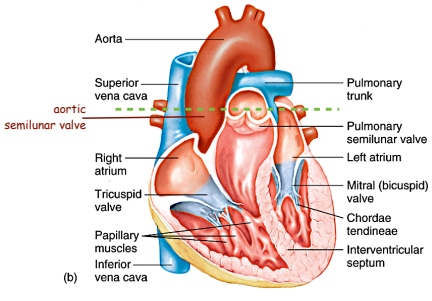
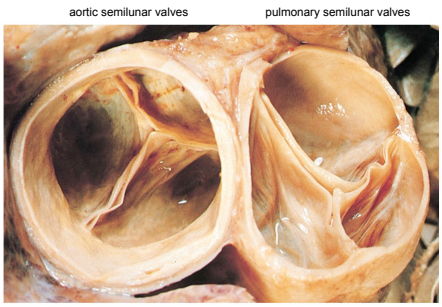


Figure 13.11



Igaku-Shoin, Ltd., Tokyo, Japan

Figure 13.12

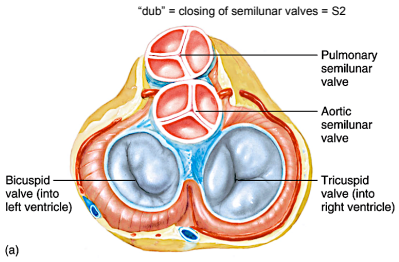


Figure 13.11

The Cardiac Cycle

Diastole
chambers are relaxed, blood can flow in

Atrial Systole
atria contract, pushing blood into ventricles

Ventricular Systole
ventricles contract with high pressure, pushing blood into the lungs and systemic circulation

Diastolic pressure (bottom number)
arterial pressure when ventricle is relaxed

Systolic pressure (top number)
arterial pressure when ventricle contracts and pumps

http://www.hhmi.org/biointeractive/cardiovascular/animations.html

lower lights

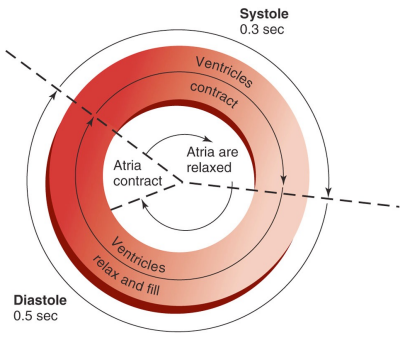
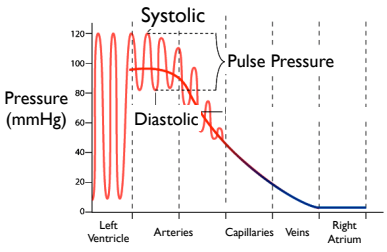


Figure 13.16

Blood Pressure:
Arterial Pressure > Venous Pressure



http://www.adinstruments.com

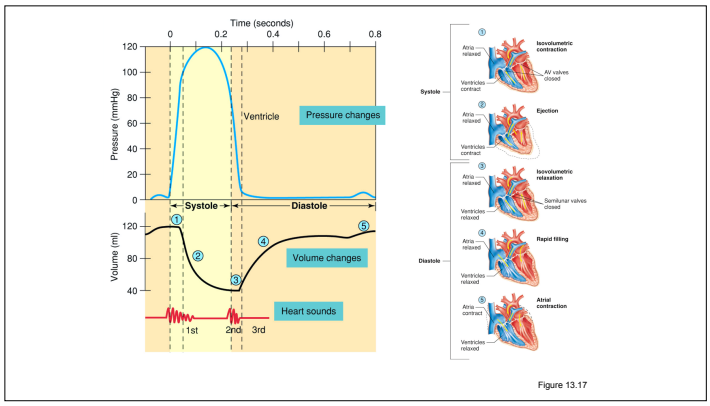


Figure 13.17

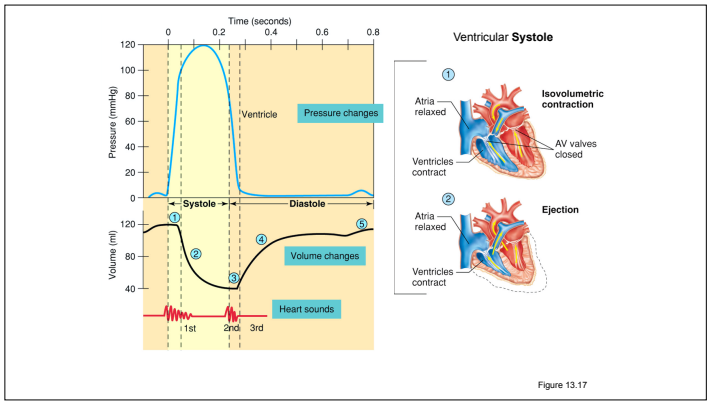


Figure 13.17

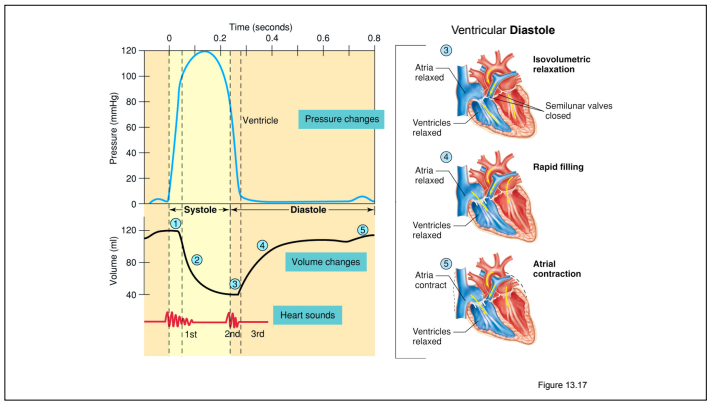


Figure 13.17

Events of the Cardiac Cycle

Atria	AV Valves	Ventricles	Semi-Lunar Valves	Blood Flow
diastole	open	**diastrole**	closed	into atria (from vena cava, lungs)
systole	open	**diastole**	closed	into ventricles
	"lub" ↓			
diastole	closed	**systole**	open	into lungs, aorta
			"dub" ↓	
diastole	open	**diastrole**	closed	into atria (from vena cava, lungs)

diastole = relaxed, systole = contracting

Heart Beat

1. Generate rhythmic stimulation to start cardiac action potential

2. Action potential will cause contraction of cardiac muscle

3. Allow action potential to spread across the heart; introduce delay between atria and ventricles so they don't contract at the same time

Contraction of Cardiac Muscle (Myocardium)

Action Potential starts from pacemaker cells in **sinoatrial node** (SA node)
- HCN channels open when hyperpolarized (**H**yperpolarization-activated **C**yclic **N**ucleotide-gated channels)
- -> spontaneous depolarization of pacemaker cells to -40 mV
- -> opening of voltage-gated Ca++ channels
- -> + 20 mV -> action potential across myocardium

Myocardial Action Potential is longer than neural action potential:
- fast Na+ channels -> fast depolarization
- slow Ca++ channels -> plateau phase
- voltage-gated K+ channels cause repolarization

Myocardium forms a functional **syncitium**, via **gap junctions**

Myocardial Contraction
Voltage-gated Na+ channels open, causing depolarization
Voltage-gated Ca++ channels open in transverse tubules
Influx of Ca++ causes release of Ca++ from sarcoplasmic reticulum
Ca++ binds to troponin to allow contraction
Ca++ ATPase pump returns Ca++ into sarcoplasmic reticulum
Na+/Ca++ exchanger pumps Ca++ from cytoplasm into extracellular fluid

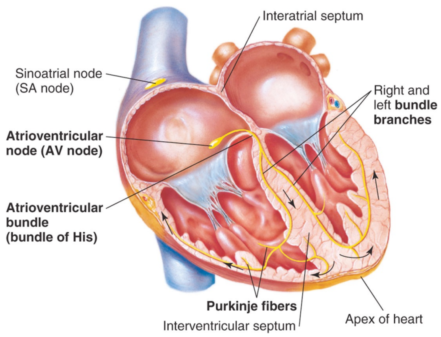
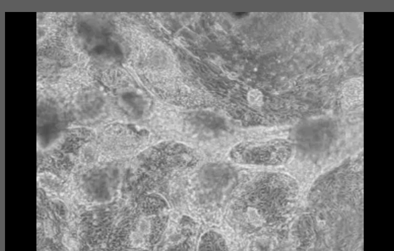


Figure 13.20

Stem cells differentiate into cardiac pacemaker cells
in vitro (in a petri dish)



Drexel University

http://io9.com/5882963/whoa-a-petri-dish-that-has-a-pulse

https://www.youtube.com/watch?v=BqzW9Jq-OVA

Spontaneous Depolarization of Pacemaker Cells

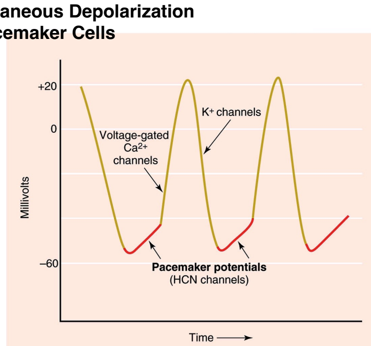
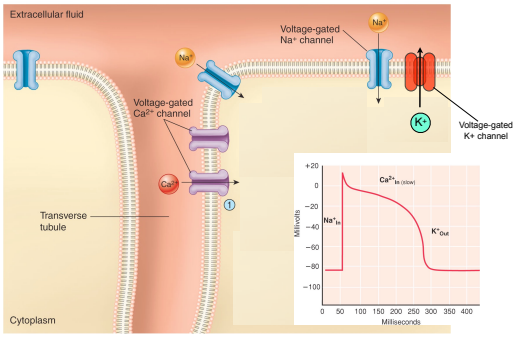


Figure 13.18



frog heart does not beat in Ca++ free buffer

Comparison of Skeletal Muscle and Cardiac Muscle

Skeletal Muscle	Cardiac Muscle
Striated; actin and myosin arranged in sarcomeres	Striated; actin and myosin arranged in sarcomeres
Well-developed sarcoplasmic reticulum and transverse tubules	Moderately developed sarcoplasmic reticulum and transverse tubules
Contains troponin in the thin filaments	Contains troponin in the thin filaments
Ca^{2+} released into cytoplasm from sarcoplasmic reticulum	Ca^{2+} enters cytoplasm from sarcoplasmic reticulum and extracellular fluid
Cannot contract without nerve stimulation; denervation results in muscle atrophy	Can contract without nerve stimulation; action potentials originate in pacemaker cells of heart
Muscle fibers stimulated independently; no gap junctions	Gap junctions present as intercalated discs

myocardial infarction (tissue damage due to lack of oxygen)
-> cardiac troponin in the blood

Table 12.8

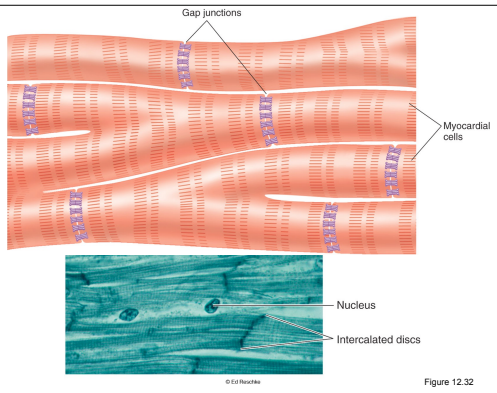


Figure 12.32

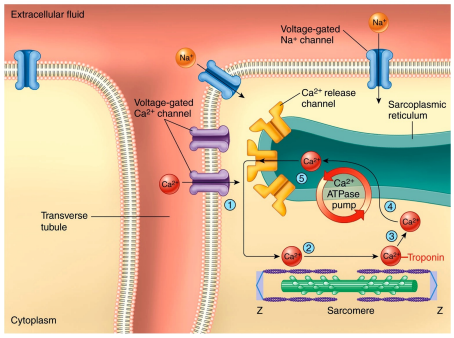


Figure 12.34

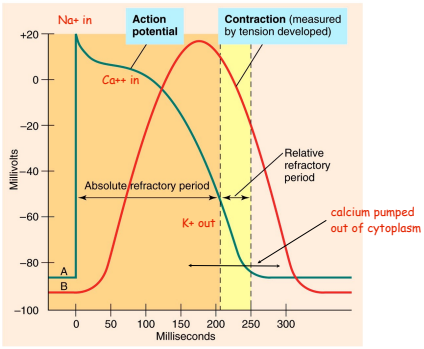


Figure 13.21

Conduction of action potential across heart and Electrocardiogram (ECG)

Action Potential (AP) spreads from pacemaker cells in **SA node**.

Myocardium forms a functional **syncitium**, via **gap junctions**

AP spreads rapidly across atria to cause depolarization and atrial systole (contraction). **[P wave]**

AP cannot cross directly to ventricles: must pass through **atrioventricular node** (AV node).

Slow conduction through AV node causes delay between atrial and ventricular contraction.

AP spreads from AV node through bundle of His and along Purkinje fibers in the walls of the ventricles. [Atria repolarize.]

Ventricles depolarize and contract. **[QRS wave]**

Ventricles repolarize. **[T wave]**

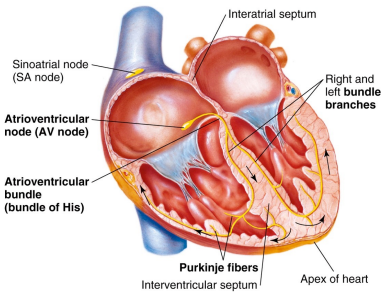
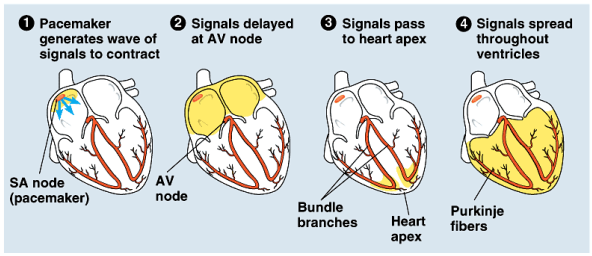


Figure 13.20

Figure 42.7 The control of heart rhythm



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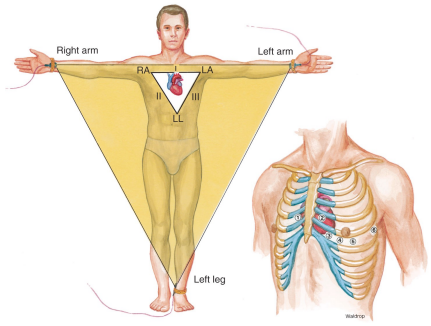
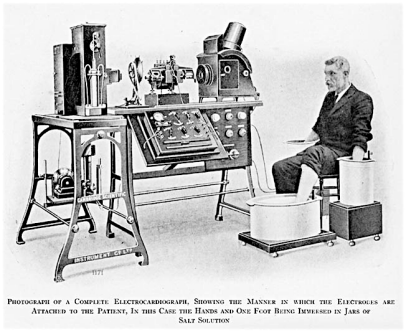
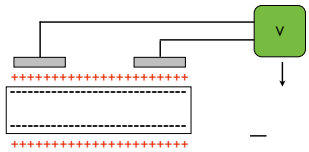


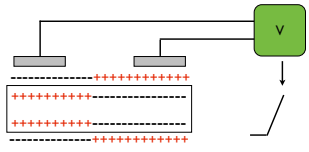


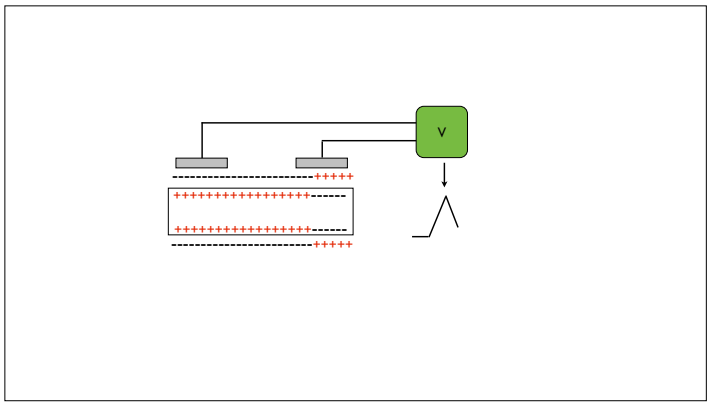
Photograph of a Complete Electrocardiograph, Showing the Manner in which the Electrodes are Attached to the Patient, In this Case the Hands and One Foot Being Immersed in Jars of Salt Solution

1911

http://en.wikipedia.org/wiki/Image:Willem_Einthoven_ECG.jpg



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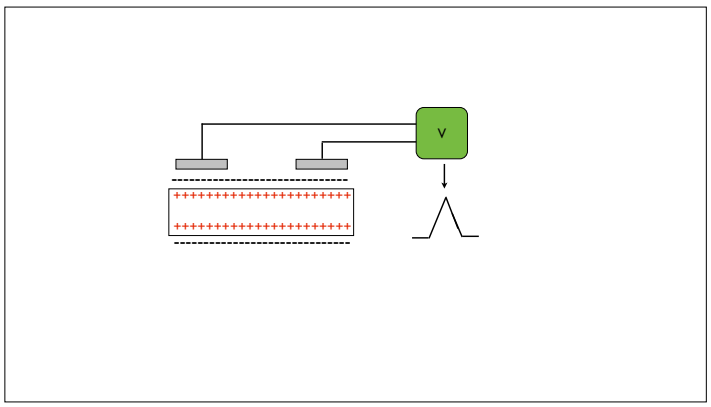
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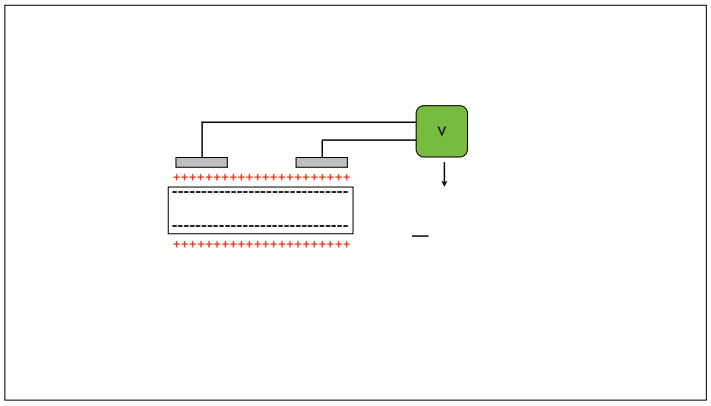
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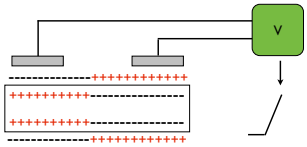
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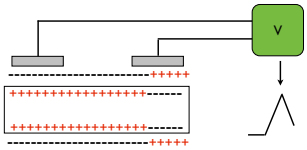
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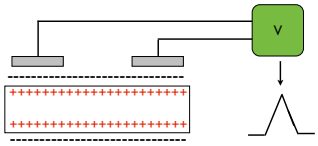
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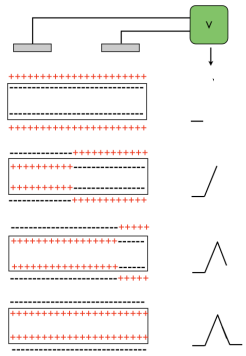
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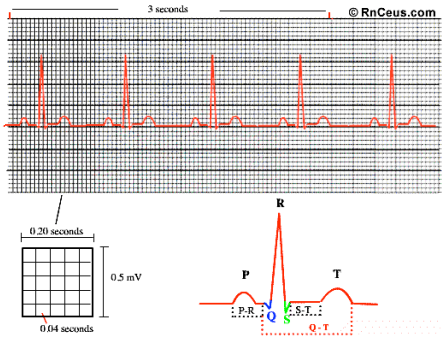
3 seconds
© RnCeus.com
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R
P
T
P-R
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S-T
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http://www.rnceus.com/ekg/ekgbasics.html

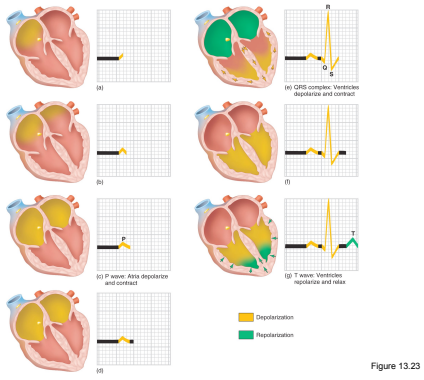
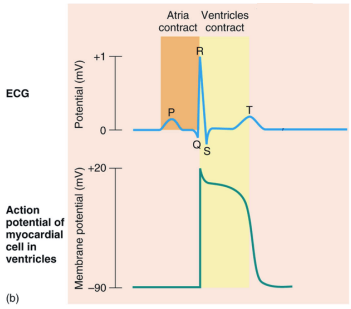

(a)
(b)
(c) P wave: Atria depolarize and contract
(d)
R
Q
S
(e) QRS complex: Ventricles depolarize and contract
(f)
T
(g) T wave: Ventricles repolarize and relax
Depolarization
Repolarization

Figure 13.23



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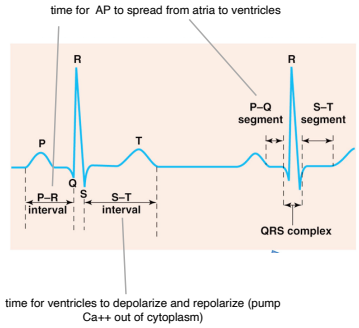
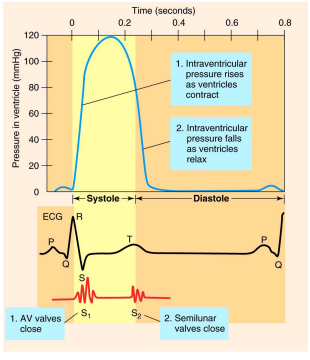


Figure 13.22

Figure 13.25



Events of the Cardiac Cycle

Electrical	ECG	Atria	AV Valves	Ventricles	Semi-Lunar Valves	Blood Flow
between beats		diastole	open	diastrole	closed	into atria (from vena cava, lungs)
SA node fires, spreads to AV node	P	systole	open	diastole	closed	into ventricles
spreads down bundle of His to Apex		systole	open	diastole	closed	
			"lub"			
spreads thru Purkinje fibers	QRS	diastole	closed	systole	open	into lungs, aorta
					"dub"	
between beats	T	diastole	open	diastrole	closed	into atria (from vena cava, lungs)

diastole = relaxed, systole = contracting

Arrhythmias and the ECG

Sinus bradycardia & tachycardia: changes in heart rate due to changes in SA Node pacemaker, e.g. by sympathetic stimulation of HCN channels -> tachycardia during "fight or flight"

Ectopic pacemakers outside of the SA node, e.g. ectopic pacemakers in ventricles -> ventricular tachycardia -> ventricular fibrillation

Circus rhythms: continuous recycling of AP around ventricles when refractory periods of myocardial cells become desynchronized. Circus rhythms can arise after damage to heart leading to disconnected portions of ventricle, or by electrical shock in middle of T wave.

Defibrillator: electrical shock depolarizes all of the myocardial cells at the same time, and syncrhonize in refractory state so SA node can restart contraction.

AV node block: damage to AV node slows or blocks atrial pacemaker signal. Can observe P-waves (of atrial contraction) without QRS wave (of ventricular contraction).

bradycardia = slow heart beat; tachycardia = rapid heart beat
fibrillation = "quivering" of myocardium; uncoordinated contraction
arrhythmia = loss of rhythm

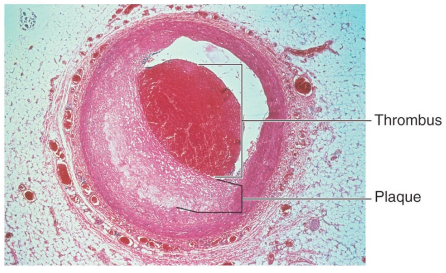


Figure 13.31a

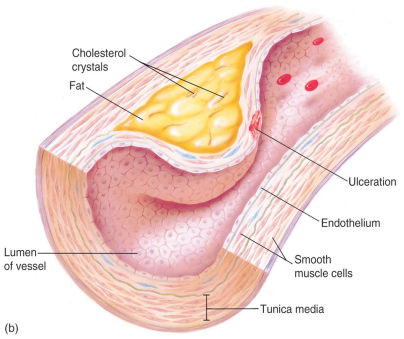
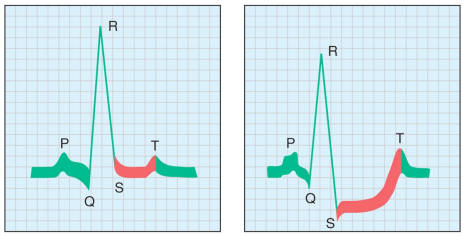


Figure 13.31b

Depression of ST interval = damage to myocardium, intracellular accumulation of Ca++



ischemia = loss of oxygen due to reduced blood flow -> cell damage & death (infarction)

Figure 13.33

bradycardia = slow heart beat
tachycardia = rapid heart beat
fibrillation = "quivering" of myocardium; uncoordinated contraction
arrhythmia = loss of rhythm

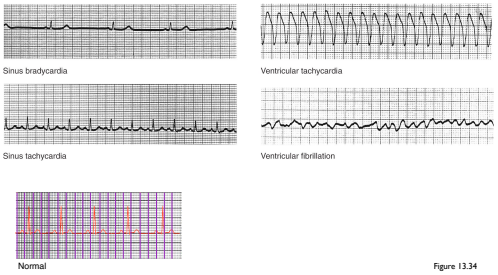
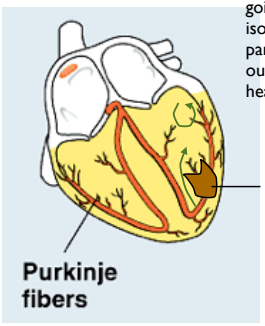


Figure 13.34

Circus Rhythms:

because of delay of AP going around damage, or isolation due to damage, parts of ventricle contract out of step with rest of heart



AV Node Block:

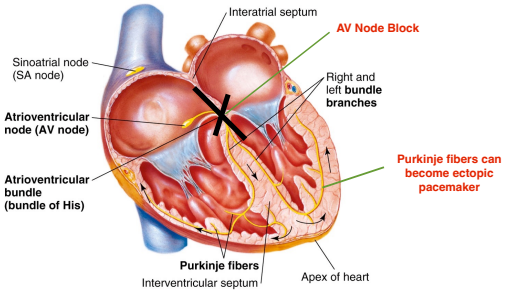


Figure 13.20

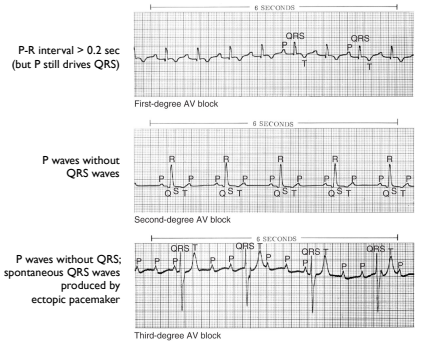


Figure 13.35

Pharmacological Regulation of Heart Rate

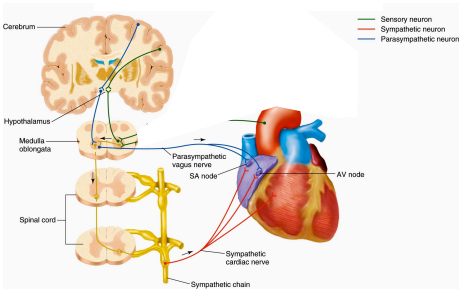
Autonomic Nervous System:

Sympathetic NS raises HR.
Sympathetic chain ganglia -> norepinephrine release onto pacemaker cells -> beta-adrenergic receptors -> increased cAMP -> open HCN channels, open Ca^{++} channels.

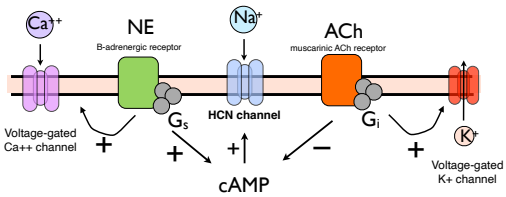
Parasympathetic NS slows HR.
Vagus nerve -> acetylcholine release onto pacemaker cells -> muscarinic receptors -> decreased cAMP -> close HCN channels, open K+ channels

Drugs to treat Elevated Heart Rate (tachycardia)
Lidocaine - blocks voltage-gated Na^{+} channels
Propranolol - "beta blocker": blocks norepi from binding beta-adrenergic receptors
Verapamil - blocks the voltage-gated Ca^{++} channels

Autonomic Nervous System & Heart Rate



Autonomic Nervous System & Heart Rate



What would the effects of NE and ACh agonists & antagonists be on heart rate?

Effect of Norepinephrine on Pacemaker Cells

HCN channels open faster & Ca++ channels open faster -> faster beat

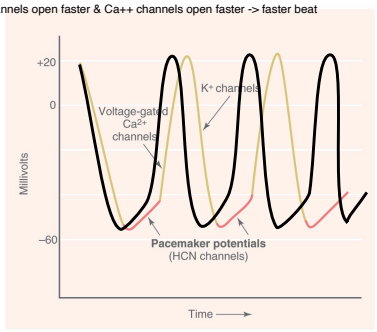


Figure 13.18

Effect of Acetylcholine on Pacemaker Cells

HCN channels open slower, more K+ channels open -> slower beat

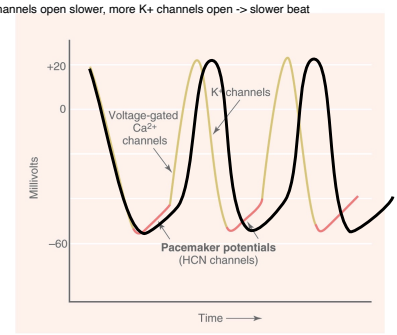
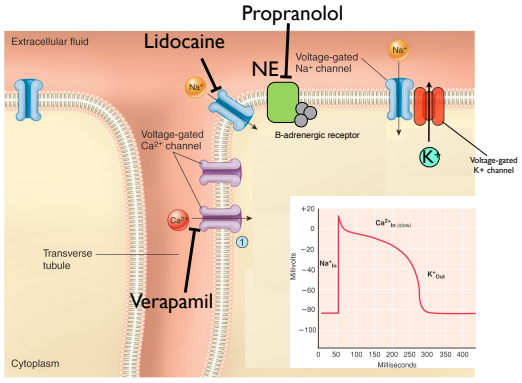


Figure 13.18



Blood Vessels

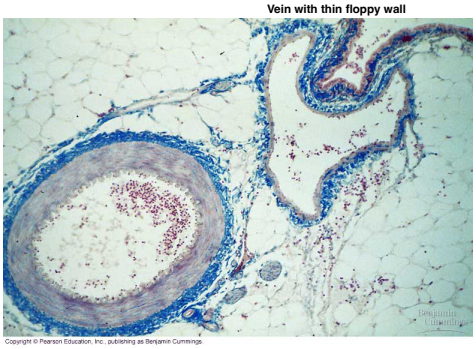
Arteries (arterial blood)
vessels carrying blood from heart towards the capillaries. Thick muscular walls to keep pressure up. High in oxygen (except for pulmonary arteries).

Veins (venous blood)
vessels carrying blood from capillaries back to heart. Very thin flabby walls with low pressure, but have one-way valves to prevent blood from backing up. Low in oxygen (except for pulmonary veins).

Capillaries
very small vessels (1 blood cell wide, with endothelium 1 cell thick) that perfuse all the tissues. Most capillaries are **fenestrated**, or have channels, or have discontinuous endothelium so that dissolved molecules can easily pass from blood to interstitial fluid & tissues. (Except for **blood brain barrier** formed by tight junctions of the capillary endothelium in brain.)

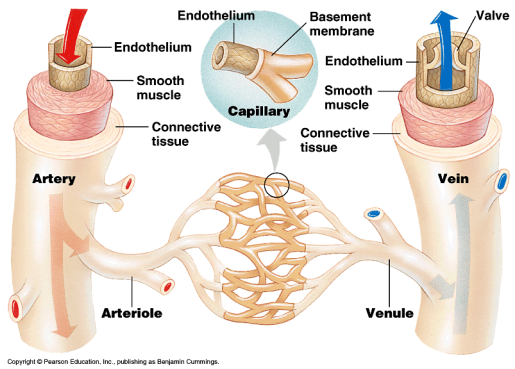
Precapillary Sphincters can regulate blood flow to capillaries or bypass thru shunt.

Vein with thin floppy wall



Artery with thick, muscular elastic wall

Figure 42.8 The structure of blood vessels



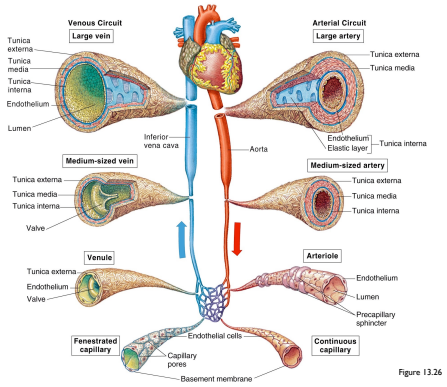


Figure 13.26

Veins: one-way valves, blood moved by skeletal muscle contraction

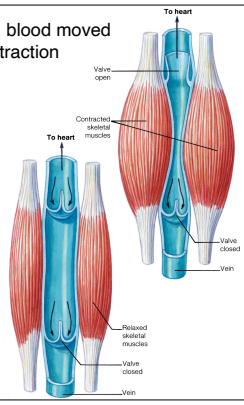
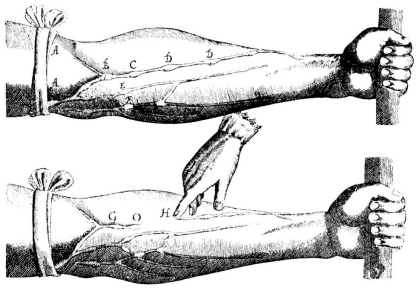


Figure 13.29

Figure 13.30



After William Harvey, On the Motion of the Heart and Blood in Animals, 1628

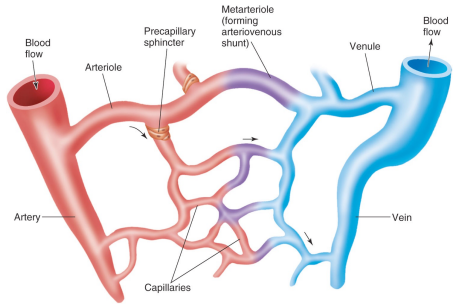


Figure 13.27

Figure 42.12 Blood flow in capillary beds

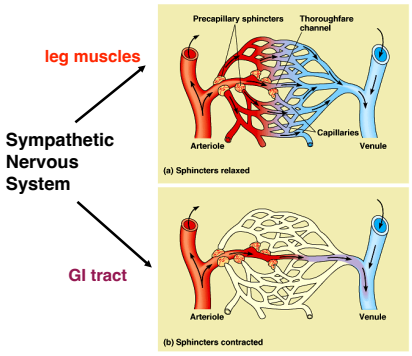


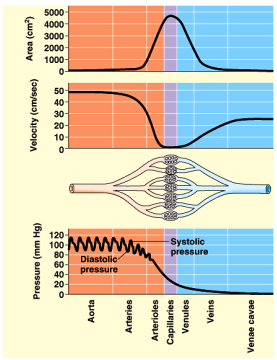
Table 13.8 | Characteristics of the Vascular Supply to the Mesenteries in a Dog

Kind of Vessels	Diameter (mm)	Number	Total Cross-Sectional Area (cm^2)	Length (cm)	Total Volume (cm^3)
Aorta	10	1	0.8	40	30
Large arteries	3	40	3.0	20	60
Main artery branches	1	600	5.0	10	50
Terminal branches	.06	1,800	5.0	1	25
Arterioles	0.02	40,000,000	125	0.2	25
Capillaries	0.008	1,200,000,000	600	0.1	60
Venules	0.03	80,000,000	570	0.2	110
Terminal veins	1.5	1,800	30	1	30
Main venous branches	2.4	600	27	10	270
Large veins	6.0	40	11	20	220
Vena cava	12.5	1	1.2	40	50
					930

Note: The pattern of vascular supply is similar in dogs and humans.
Source: Animal Physiology, 4th ed. by Gordon et al., © 1982. Adapted by permission of Prentice-Hall, Inc., Upper Saddle River, NJ.

Table 13.8

Figure 42.10 The interrelationship of blood flow velocity, cross-sectional area of blood vessels, and blood pressure



Endothelial cell lining capillary

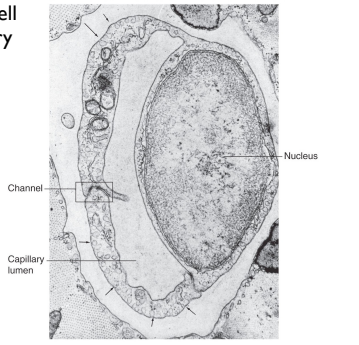


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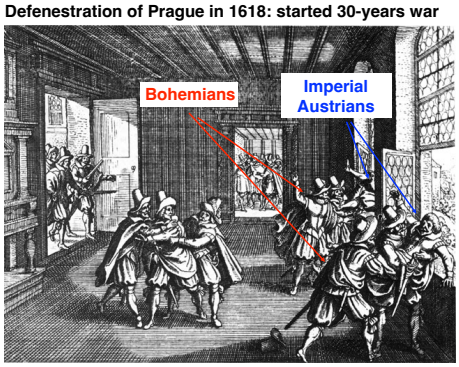
Figure 13.28

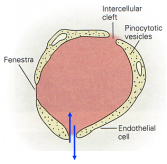
Defenestration of Prague in 1618: started 30-years war



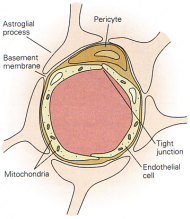
Fenestrated Capillaries vs. Blood Brain Barrier

General capillary

Periphery of Body



Brain capillary



Brain

Only transported or hydrophobic molecules can cross

end recording